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General Medicine

STUDY OF CLINICAL PROFILE AND PROGNOSIS OF PATIENTS WITH PARAQUAT POISONING AT TERTIARY CARE HOSPITAL SULLIA, DAKSHINA KANNADA,KARNATAKA, INDIA

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ABSTRACT

Background: In Indian market paraquat is easily available for agricultural use. Several options studies have been done on paraquat poisoning globally. However, only few studies on paraquat poisoning are done in India. Considering it high mortality associated with paraquat poisoning we need to study extensively and need to develop better treatment options. It is important to make public aware about the poison and its fatality.

Aims & Objectives: To study the clinical features, laboratory parameters, treatment and outcome of paraquat poisoning patient admitted at KVG Medical College, Sullia.

Materials and methods: It is a cross sectional study carried out at KVG Medical College, Sullia, Dakshina Kannada, Karnataka during June 2019 to June 2020. Patients admitted to hospital with paraquat poisoning above 18 year of age were enrolled for the study.

Results: Of 10 patients, 7 were male and 3 were female. 2 patients survived and 8 patients died. Most of the patients died because of acute respiratory failure and acute kidney injury.

Conclusion :Study showed very high mortality in paraquat poisoning. Complications were observed in all patients including survivors even after treatment.

KEYWORDS : Paraquat, High mortality, ARDS

INTRODUCTION

Paraquat ingestion is a leading cause of fatal poisoning in many parts of Asia, Pacific nations, and the Americas[1]. Paraquat is a rapidly acting, non-selective quaternary nitrogen herbicide that is relatively inexpensive. These characteristics contribute to its widespread use in the rural areas of developing countries where it remains readily available and it is a common method for intentional self-poisoning[1]. In plant study paraquat exerts its herbicidal activity by inhibiting reduction of Nicotinamide Adenine Dinucleotide Phosphate (NADP) to it's reduced form NADP Hydride (NADPH) during photosynthesis which leads to the formation of superoxide anion, singlet oxygen, and hydroxyl and peroxyl radicals, these reactive oxygen species (ROS) interact with the unsaturated lipids of membranes, resulting in the destruction of plant organelles, inevitably leading to cell death[2].

Increased mortality seen in paraquat poisoning is either due to significant lung injury or multiorgan failure[3]. The case fatality rate in paraquat is as high as 70%[4]. Paraquat concentration in the lung is 10-15 times greater than in the plasma due to the energy-dependent uptake of the poison by the alveoli. This gradient persists despite decreasing paraquat levels in the blood. Involvement of the lung in the form of diffuse alveolitis (over 1–2 days) and subsequent pulmonary fibrosis is the hallmark of paraquat poisoning and Acute Respiratory Distress Syndrome (ARDS) sets in after 24–48 hours of exposure[5]. In majority of patients features of early ARDS occurred within 24 hr of consumption of paraquat. Apart from that poisoning with paraquat leads to both local and systemic effects.

Renal failure in paraquat poisoning is multifactorial due to hypovolemia, circulatory failure, septicaemia, and direct toxicity related to redox cycling[6]. It has been suggested that the mechanism involved in multiple organ damage is primarily related to oxidative damage, reactive oxygen species, immune activation and inflammatory mediators[7].

In the Indian market paraquat is available as a liquid concentrate (29.1%) for agricultural use. Several studies have been done on paraquat poisoning globally however, only few studies are done in India.

Considering high mortality associated with paraquat poisoning, we need to study in detail and need to develop better treatment options. It is important to make public aware about the poison and its fatality.

AIMS & OBJECTIVES:

To study the clinical features, laboratory parameters, and outcome of paraquat poisoning patients admitted in KVG Medical College, Sullia, Karnataka.

MATERIALS AND METHODS

It is a cross sectional study carried out at KVG Medical College, Sullia, Dakshina Kannada, Karnataka. Patients admitted during June 2019 to June 2020 with paraquat poisoning were enrolled into the study.

In this study we studied the clinical features at the time of presentation, treatment details and outcome of 10 cases who were admitted at our hospital with paraquat poisoning.

Detailed history of patient, time from ingestion to hospitalization, initial management at other hospitals prior to referral has been recorded. Amount of paraquat ingested was classified as mild (<10 mL), moderate (10–20 mL), and severe (>20 mL)[8].

Lung involvement was diagnosed based on hypoxemia and infiltrates on chest X-ray, patient having serum creatinine ≥1.5 mg/dl were classified to have Acute Kidney Injury. Those with elevated liver enzymes and serum bilirubin were classified to have acute liver injury.

INCLUSION CRITERIA

All the patients admitted with paraquat ingestion above 18 year of age were included in the study.

RESULTS

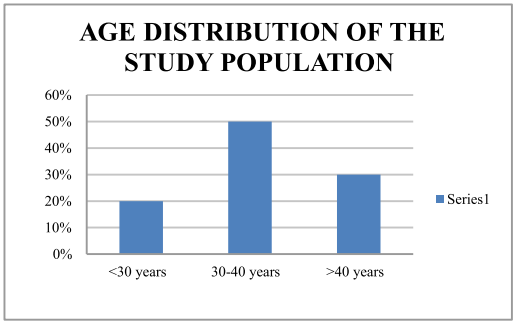

Figure 1: Age distribution of study population

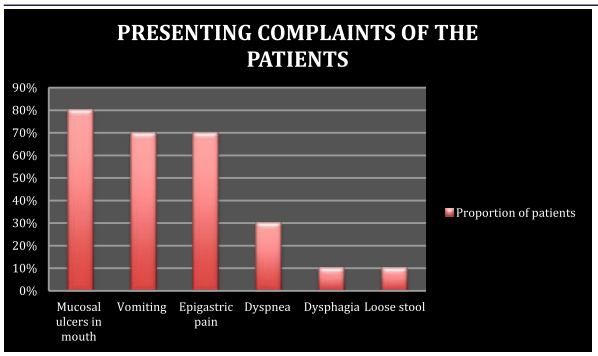

Figure 2: Proportion of patients with their presenting complaints.

Table 1:Time from ingestion to hospital contact and amount consumed.

Ingestion to hospital contact time	
<6 hours	6 (60%)
6-24 hours	3 (30%)
>24 hours	1(10%)
Amount consumed	
Mild (Less than 10 ml)	3(30%)
Moderate (10 to 20 ml)	1(10%)
Severe (More than 20 ml)	6(60%)

Table2:Complications and laboratory findings

Lung Injury	8(80%)
Acute kidney injury	8(80%)
Hypotension	3(30%)
Important Lab parameters	
Serum creatinine > 1.5mg/dl	8(80%)
Total bilirubin >2mg/dl	1 (10%)
Alkaline phosphatase >200IU/L	2(20%)
Aspartate Aminotransferase >60U/L	1(10%)
Alanine transaminase > 70U/L	1(10%)
Serum albumin < 3.5g/dl	1(10%)
Serum potassium >5.5mEq/dl	8(80%)
Total Creatine phosphokinase >500U/L	7(70%)

Among 10 patients, 70% were males and 30% were females and the median age was 38 years. 6 patients presented within 6 hours of ingestion of paraquat and 60% had consumed amount more than 20 ml. At the time of presentation 80% patients had mucosal ulcers in oral cavity, 70% had vomiting, 70% had epigastric pain, 30% had dyspnea, 10% patient Dysphagia and 10% patients had loose stools**(Figure 2)**. Lung injury were documented in 80% patients and all of them needed mechanical ventilation. Hypotension was documented in 30% patients. 80% patients had acute kidney injury.

Bilirubin was marginally increased in 10% patients, while aspartate and alanine aminotransferases were elevated more than 1.5 times the normal was noted in 10% patient. Serum albumin were low in 10% patients, serum creatinine was raised in 80% of patients, in 80% of the patients serum Potassium was raised more than 5.5 mEq/L and in 70% patients total Creatine phosphokinase (CPK) was raised **(Table 2)**.

Gastric lavage and activated charcoal were given to almost all patients at emergency department. All patients had received steroids-Methylprednisolone 7 patients and Dexamethasone intravenous 3 patients. Cyclophosphamide intravenously given for 1–2 days along with steroids in 30% patients, 30 % patient underwent dialysis. Apart from this N-Acetyl-Cysteine and Vitamin C injections and other supportive treatment were given to all patients.

OUTCOME

During course in the hospital, 2 patients survived while 8 patients died within 3 days of hospitals admission.6 out of 8 patients died were under the influence of alcohol at the time of ingestion of paraquat and it is observed that these patients died earlier as compared to remaining 2 patients. Among survivors, all of them had ingested mild amount around 2-3 ml and they presented within 30 minutes of ingestion.

Most common cause of death in patients was acute lung injury and kidney injury. In current study whoever developed respiratory complications in the form ARDS and AKI with hyperkalemia all of them died.

DISCUSSION

In this present study critical care team were able to identify the compound as all patients relatives had brought bottle/label with them. Initial symptoms of vomiting, epigastric pain, oral mucosal ulcers noted in most of the patients, some patients developed dysphagia and dyspnea later. Altered sensorium was seen in 2 patients. However A.Bhalla et al did study at tertiary care center of Northern India in 2008, they studied 17 patients in case series and they found most common symptoms as vomiting(100%), followed by altered sensorium (59%), oral ulceration or dysphagia (53%), dyspnea (41%), or loose stools (24%)[9].

Present study showed there is no relation between clinical features at the time of presentation and final outcome. However, patients who ingested less quantity and presented early to hospital showed favoraable outcome similar to study done by Sandhu J at al in Panjab on 17 patients in 2003, they also found out that survivors were mainly the patients who had consumed small amount of paraquat and had received early and effective treatment[10].

Gastric lavage and activated charcoal were given to almost all patients at emergency. Removal of ingested paraquat immediately by inducing emesis or by gastric lavage in a health care facility is essential step, activated charcoal is effective adsorbent, administration of repeated doses of 60gm of activated charcoal by gastric tube every two hours (total three to four doses) is useful and showed benefit in current study similar to other studies[11,12].

At present, oxygen is not a part of treatment, because too much oxygen can rapidly promote pulmonary changes through oxidation[7]. In present study oxygen was not given until they developed desaturation. Immunosuppression with combination of cyclophosphamide and methylprednisolone was shown to be beneficial in moderate-to-severe cases by prevention of ongoing inflammation this is similar to study done by We WP et al during the period of January 1,1997 and December 31, 2009 on 1811 patients in Taiwan[12].

Paraquat is excreted primarily by kidneys, and therefore, hemoperfusion has often been indicated as an appropriate step for treatment and is considered 4-6 times more effective than hemodialysis[13]. In current study hemodialysis was carried out in 3 patients who presented early , among 3 patients who underwent hemodialysis 2 patient survived but both patients who survived had ingested very mild quantity of paraquat about 2- 3 ml.

Even superoxide dismutase, vitamins C and E, N-Acetylcysteine, desferrioxamine has been tried in treatment of paraquat poisoning but none of them showed complete benefit in systematic review carried out by M. Eddlestone et al in 2002[14]. In current study also Vitamin C , N-Acetylcysteine didn't show much benefit.

In this study majority of the patients presenting with paraquat consumption had taken 20 % paraquat, those who had taken less quantity (less than 20ml) survived for longer time in comparison to the patients with large amount (more than 20 ml) of paraquat intake, however complications had set in almost all cases including the survivors suggesting even low dose is harmful.

CONCLUSION

Paraquat poisoning is dangerous with high mortality and there is no antidote available. Current study showed very high mortality and all the patients whoever developed respiratory involvement (ARDS) died inspite of all possible efforts.

Early deaths were noted in patients who ingested paraquat with alcohol that warrants further study.

Public health education is required to increase the awareness about paraquat poisoning and its high mortality. Early decontamination, early use of immunosuppressive drugs and hemoperfusion or hemodialysis may prevent death.

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